

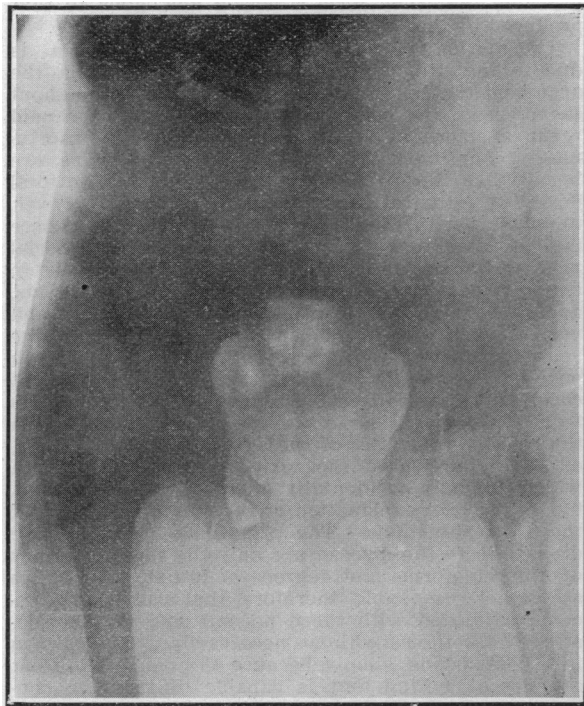
1902, by Dr. Lorenz. Reposition was easily made, and the doctor pronounced stability fair. Temperature did not run over 99.5° F.; left hospital in one week, returning to Arizona. Cast in second position applied at end of eight months. Cast in third position three months later; after remaining in this position three months, the cast was permanently removed.

Child is three pounds under weight, one inch under size; middle of thigh measuring 3 cm. less than the right, and 1.5 cm. shorter. There is a decided thickening about the joint, which is firm and resisting. The child walks with a limp, and has a decided scoliosis, left lumbar and right dorsal. The action of the adductors is entirely lost. Result may not be anatomical, but it is much better than a transposition.

Case No. 2.—T. B., first child; father 59, mother 40; prolonged labor; vertex presentation; mother did not feel motion of child during period of gestation; patient thrived but had no natural movement of limbs or head for twelve months. At the age of 2 years, mother observed right leg shorter than other. At 3 years and until date of operation, the patient walked with the assistance of one hand.

Examination shows a very fat girl, 4 years old, four pounds over weight, one inch over size; very slow mentality; right limb measures 3 cm. short; joint very lax.

Operation by Dr. Lorenz at Medical College, November 13, 1902. Reposition readily accomplished. Left hospital at the end of one week; highest temperature 100.2°. First cast removed June 21st. Result, transposition. Patient etherized and head of femur replaced and cast applied in second position. September 9th second cast was removed and one applied in third position, which remained until December 9th, when it was permanently removed.



Head of the femur is apparently in perfect position, though child has a swinging limp. X-ray picture No. 4 taken March 15th shows anatomical result.

Case No. 3.—E. H., female, first child; 11 pounds; vertex presentation; dry birth; strong infant; walked at about 15 months, but right foot dragged and was noticeably wrong. Always well nourished, but clumsy and inclined to fall when running; has a distinct limp.

Blond girl, 6 years old, 5 pounds over weight, 1.5 inches over size, 1 inch over normal chest measurement; right limb 3 cm. short; joint very lax, marked loss of contour, affected side much more rounded.

Operation November 13, 1902, Medical College, by Dr. Lorenz. Reposition readily produced. Left hospital at the end of one week. Highest temperature 100.6°. Came back June 29th, cast removed and limb placed in second position because of slight antversion of head of femur; limb was rotated inward and a cast applied which extended below the knee. September 16th, when this cast was removed, the head was found in good position, and a cast in third position was put on. This remained on two months, and at the end of that time was permanently taken off.

The patient shows a slight right lumbar and left dorsal scoliosis; the adductor group is almost entirely destroyed; patient limps a little, but judging from the X-ray picture should pronounce it an anatomical result.

## THE ETIOLOGY OF INFANTILE ECLAMPSIA.\*

By JAMES M. FRENCH, M. D., San Diego.

THE term infantile eclampsia is used to designate a type of convulsive seizure, rather than a condition peculiar to infancy. Several writers, indeed, have given to the word "infant" in this connection a breadth of meaning even greater than that which it has in law and refer to the occurrence of the same kind of convulsions in adults. For the present, however, I desire to consider the subject only in its relation to infants and young children. The term "convulsions of children" is to be preferred.

No definition of these convulsions is complete which does not recognize their similarity to those of epilepsy. The seizures are often identical, yet I do not believe that there is necessarily more than a resemblance. This is, however, one of the questions on which I hope to hear an expression of the opinion of other members of the society. I have elsewhere defined infantile eclampsia as "convulsive seizures like those of epilepsy, occurring during infancy and early childhood, generally as a result of toxemia or reflex irritation, and sometimes developing into epilepsy." It is a neurosis, not an organic disease; it can hardly be called a symptom, for it has more of the nature of an accident that may occur at the onset of many diseases or interrupt their course.

In studying the etiology, there are for consideration an inherited or acquired predisposition and an immediate, exciting cause. First among predisposing influences, we have the abnormal excitability or instability of the nervous system which is often recognizable as a family peculiarity. Some writers, however, regard this as an evidence of more than usual weakness in the control exercised by the inexperienced and untaught cerebral centers over those of the spinal cord during the first years of life. The predisposition is to be regarded as inherited when there is a history of similar convulsions, or of insanity, epilepsy, hysteria, or other neurotic disease in other members of the family, more particularly if it has been prominent in the parents or grandparents, and when one or both parents are drunkards. The children thus predisposed are most liable to become confirmed epileptics. Lancereaux was one of the first to recognize the influence of alcoholism, but it is perhaps not sufficiently emphasized at the present time. As a result of it we see not only an entire family of children afflicted with convulsive weakness of the nervous system, but it is sometimes possible to pick out the children that have been begotten after the beginning of a dissipated life or during periods of drunkenness. Periodical drunkenness in the parents, I believe, accounts for many of the instances in which only certain members of the family suffer from convulsions or become epileptic. A history of tuberculosis, diabetes, or syphilis in the ancestry is also considered a possible source of abnormal nervous irritability.

Another important predisposing influence is malnutrition. It is perhaps, in part at least, as a result of malnutrition that rachitic children are predisposed to general convulsions as well as to laryngismus stridulus and other localized spasms that do not strictly belong to the class under consideration. Possibly, however, the pressure of the deformed cranial bones may have its influence. If so, the seizures are induced in much the same manner as those of epilepsy arising from the growth of gummata or exostoses within the cranium. Gowers, however, believes that there is in rachitic children an "excessive activity of the centers of the brain and cord on which reflex spasm and convulsions depend." The infant that is feeble from birth, the one with congenital syphilis, the hydrocephalic infant, all these are similarly predisposed.

But although a large ratio of the children affected with infantile eclampsia exhibit a predisposition,

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there is no good ground for the assertion that an abnormal instability of the nervous system must exist in order to render such seizures possible. The most vigorous child, free from discoverable hereditary taint, may be overwhelmed by either a chemical or a mechanical irritant; but such children rarely have a succession of convulsions. Gee reports 56 cases in a total of 102 in which no predisposition was discoverable. There can be no doubt, however, that the existence of a predisposition renders any of the exciting causes more effective, and it is for this reason that the most trivial irritation will sometimes produce a seizure or a series of convulsions.

Most authors state that boys are more susceptible than girls, although in epilepsy women are somewhat more frequently affected. In my own experience, sex has not appeared to exert any influence. Nationality has its bearing on many cases; the Latin races are unquestionably more susceptible to convulsive seizures than are any others.

There are a great many exciting causes; so many indeed that there is no longer any excuse for the consideration of an idiopathic origin. It is a no more plausible explanation of a case than was the more ancient doctrine that the seizures were the work of evil spirits.

Cases not infrequently occur in practice for which it is exceedingly difficult, if not impossible, to give a satisfactory explanation and in such cases it is generally better to wait a few days for further developments than to hazard an improbable guess.

A good deal has been written about misguided waves of nervous influence, katabolic discharges of nerve cells and other unknowable forces as the immediate source of the convulsive movements. The more recent theory of neuron action helps us to understand the possibility of such discharges, it is true, but for the present comparatively little can be gained from attempts to further elucidate the actual occurrences outside the physiological laboratory. It is noteworthy, however, that Berg, in a recent article, advocates the theory advanced by Schroeder van der Kolk, that the accumulation of carbon dioxide in the blood, following the initial arrest of respiration, is the immediate exciting cause of the convulsive movements.

The exciting causes belong for the most part to one or other of two classes, namely, the direct, or symptomatic, and the reflex. The former class includes all those cases in which the stimulus acts directly upon the nerve centers. The most important stimuli acting thus are fever and toxemia, but fever is gradually giving place to toxemia in the numerical estimation of causes. Whether febrile or toxemic in origin, most of these seizures represent the chill of an adult, but a chill, after all, is only a modified convulsion. Before the action of the bacterial poisons was recognized, high temperature was looked upon as the origin of nearly all convulsions that occurred at the beginning of the acute infectious diseases or during their course. The only form of toxemia then recognized was that resulting from uric acid, lead, and other mineral or vegetable poisons. There are still some authorities who support the view that fever is the most important convulsive influence, especially with reference to the paroxysms that occur during the course of a disease when the temperature rises rapidly to an unusual degree. That many of the toxins produce convulsive action has been repeatedly demonstrated, and I believe that most attacks which mark the onset of an infection are of toxemic origin, as maintained by Hensch and others. Although the temperature may be high, the spasm results from the action of the bacterial poisons on the nerve centers, chiefly those of the cerebral cortex or pons Varolii—the convulsion center of Nothnagel. It is thus a result of the same cause that produces the fever. This view is supported by the fact that the time of the convulsion does not always coincide with that

of the greatest rise of temperature. In measles, smallpox, and sometimes in scarlet fever, the spasm may occur before the temperature has reached sufficient elevation to justify the supposition that it is due entirely to heat, and the subsequent rise of temperature is often so great and so rapid as to render a repetition of the convulsion much more common than it generally is, if this were the only influence producing it. Initial convulsions are generally single, except in foudroyant cases. They are rare in pneumonia, although the fever is high, except in young infants and when the lungs are extensively involved. Steiner's suggestion that they are then a reflex result of the local irritation of the lung has not been generally accepted. It seems more probable that they arise from the bacterial toxemia, to which is added, however, a variable degree of carbon-dioxide poisoning. The onset of diphtheria is seldom characterized by a convulsion, for the toxins are rarely absorbed until later in the course of the disease, and even then the temperature is not always excessively high and it is even more rarely rapid in its rise. In malaria affecting young children, the convulsions are often exceedingly severe, although the temperature may be moderate. It is possible, of course, that the plasmodia are the direct irritant, but it seems no less probable that they form a poison analogous to that produced by the bacteria.

Those who favor the view that the eclampsia is due to high temperature urge against toxemia the argument that, as the intoxication persists throughout the disease, the convulsions arising from it should recur at more or less regular intervals. Unfortunately, our means of measuring temperature are better than those for estimating toxemia. The total degree of intoxication is generally believed, however, to coincide with the elevation of temperature. There is still much to be learned in regard to bacterial poisons, but we know that all of them are not alike in action. Many bacteria produce two or more toxins, and these may be very unlike in their action, and very unequal in their tendency to produce convulsive seizures; indeed they may not possess any such power. Much of the diversity that is seen in different cases of the same disease, and many of the departures from the normal course are due to the variations in the relative quantities of the several toxins produced, or to the action of the toxic products of bacteria which are only accidentally present. This variation, too, is the best explanation of the symptomatology of the acute infections. The symptoms by which we diagnose the disease are only the manifestations of different forms and degrees of intoxication. It is altogether reasonable, therefore, that a child may become saturated with these poisons and may be destroyed by them, without necessarily being thrown into convulsions, simply because the poisons present are not of a kind that is capable of inducing convulsions. In this light it is much easier to understand how a child may escape convulsions in the presence of profound toxemia, even if toxemia were the only possible exciting cause, than that it should escape them when the temperature becomes high, if fever alone induced them. Yet we often see hyperpyrexia without spasms.

Anemia and altered blood-states, other than those produced by the toxic substances formed by bacteria and animal parasites, are accountable for the eclampsia in some cases. This is illustrated in acute nephritis and less commonly, during childhood, at least, in diabetes and chronic nephritis. None of these conditions is attended, ordinarily, with elevation of temperature, but violent convulsions may occur. We call the condition uremia, although neither urea nor uric acid is probably the cause of its production. The ptomaines afford us another example of chemical poison which may act independently of fever, and eclampsia due to alcoholic intoxication is closely related to this class, since the poison is formed by

micro-organisms. Ptomaines are formed in food either before or after it has entered the body, and it is often difficult to distinguish the seizures induced by them from those of a reflex nature having their origin in the alimentary canal. Some writers include in the class of chemical irritants other poisons that are supposed to be produced by intestinal parasites. The anemia produced by the bothriocephalus latus and that of ankylostomiasis, for example, are caused by poisons of this character and not by the abstraction of blood, and it is probable that other parasites may form poisons capable of inducing convulsions. Here again it is difficult to exclude reflex irritation, particularly when a large coil of lumbricoid worms obstructs the lumen of the bowel.

The commonest of the reflex irritants is gastro-intestinal disturbance. When a spasm follows directly upon the ingestion of a large quantity of untainted food, it is safe to conclude that the over-distention of the stomach is the exciting cause—a distention that is increased by the secretion of a pint or more of gastric juice required in digestion. Such paroxysms are often accompanied or followed by vomiting and the character of the food can then be determined. Improper food is an equally common cause. This includes unripe fruit, the swallowing of pits with the fruit, and many coarse or indigestible substances. Even the most wholesome food in too great quantity may prove obstructive and induce a convulsion. Foreign bodies act in the same way.

The late stages of gastro-intestinal diseases in infancy, gastro-enteritis, enterocolitis, dysentery, and cholera infantum are often attended with eclampsia, but these probably belong to the class of direct irritation, the toxemia or the fever inducing the attack. It is possible, however, that they may be due to auto-intoxication, the altered blood state, or the impoverishment of the blood supply to the brain. The convulsive seizures at the beginning of acute anterior poliomyelitis are probably a reflex from the spinal irritation, for there are no recognized convulsive centers in the spinal ganglia. Those of brain tumor, cerebral hemorrhage or sclerosis, and injuries, have a definite pathology and are therefore not neuroses.

Among other forms of peripheral irritation generally referred to are dentition, phimosis, masturbation, compression of the testicle within the inguinal canal, retention of urine, cutaneous eruptions, foreign bodies in the auditory meatus or severe earache due to inflammation and such accidents as scalds and burns. With the exception, however, of those rather unusual cases in which the nature and severity of the irritation is altogether evident, none of these causes is considered, as a rule, until all other possibilities have been excluded. For my own part, I have never seen a convulsion which I felt justified in attributing solely to teething, although I have seen many children who suffered from some form of gastro-intestinal disorder with the irruption of every tooth, and it could not always be attributed fairly to overfeeding, which is so often practiced at this time in order to allay the fretfulness of the infant. Painful or irregular dentition may of course be a cause, but it must be remembered that such irregularities occur chiefly in rachitic and otherwise debilitated infants, already liable to be thrown into spasms by the most trifling disturbance. In scalds and burns there seems to be every reason to attribute the seizure to reflex irritation, and yet there is good ground for referring many cases to an influence acting directly upon the centers, for the convulsion is generally preceded by a period of increased prostration sufficient to suggest the possibility of an altered state of the blood or even an embolism.

Cerebral congestion is the only explanation of some cases, as when the paroxysm follows fright, crying, a fit of coughing in pertussis, or a violent outburst

of anger. In such cases the irritation is doubtless direct, but when, as occasionally happens, a fit of anger, grief, or other emotional disturbance in the mother so alters the character of her lacteal secretion as to induce convulsions in the infant, the influence is to be regarded as a long-ranged reflex. A thorough search for possible sources of irritation will often enable us to avoid what might later prove to be an incorrect explanation, as in a case in which, as a last resort, I had the infant stripped of its clothing and found a large area of erysipelas around a vaccination wound that was supposed to have been entirely healed.

## POSTURE IN THE TREATMENT OF DISEASE.\*

By C. M. COOPER, M. B., Ch. B., M. R. C. S., L. R. C. P.

THE influence of disease upon attitude and position in obedience to the principles enunciated particularly by Hilton has been studied to some extent; though even in this there is still much that could be added to our knowledge if to the improved armamentarium of our day could be added the discerning bedside acumen of the older clinicians. How instructive it is to note nature's endeavor to immobilize an inflamed or painful structure consisting as it does of two elements—

1. An effort independent of conscious volition as e.g., the reflex contraction of muscles in the vicinity of painful joints.

2. A conscious volitional effort as e.g., the chin-on-hand attitude adopted by one suffering from disease of the upper cervical vertebrae. The quicker our patients' reaction time the sooner would we naturally expect them to discover these relieving volitional attitudes; attitudes which we physicians, imitating that wise teacher, Nature, would adopt in treating other patients afflicted with a like disease. Moreover, when the effort involves a process of reasoning we would expect the more intellectual of our patients to adopt attitudes and positions of relief which their less intellectually favored brethren would be unable to devise and such we undoubtedly to some extent find; but we must recollect that the vast majority of our patients know nothing of either anatomy or physiology and consequently cannot be expected to deduce and assume many of these volitional positions and we must further remember that the subject is complicated, inasmuch as the active assumption and maintenance of any position involves muscular efforts which in their turn may be painful and which certainly cause changes in blood-pressure and frequency of heart-beat, which under some circumstances would be inimical to the general welfare of the patient. Hence, we must not expect too much from our patients, but must ourselves brood over the effects of active and passive postures if we desire to intelligently use the same. In the literature at my disposal I have been able to find only desultory reference to position in the treatment of disease, if we except from our discussion the treatment of injuries to bones, joints, muscles, and ligaments—hence the incompleteness of this paper must be apologized for, and as the contemplation of active postures meets with the objections previously outlined it is with passive attitudes that I expressly wish to deal.

Temporary ischemia and congestion of otherwise healthy cerebro-spinal areas seem to play quite a little part in the production of functional symptoms and moreover many evanescent symptoms occurring during the course of organic nerve diseases are undoubtedly due to temporary circulatory changes. Most often these vaso-motor changes are apparently localized to definitely small areas, yet on the other hand there are states e.g., syncope and concussion in which the whole brain appears to be imperfectly supplied with blood, and others—the so-called cerebral congestions—in which the opposite state exists. It is the common custom to find patients suffering from

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